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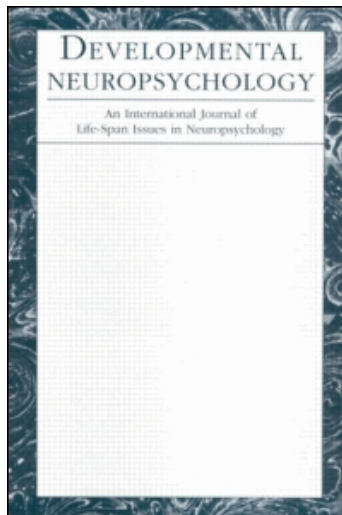
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Fronto-Central Dysfunctions in Reading Disability Depend on Subtype: Guessers but not Spellers

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The goal of this study was to test the hypothesis that the inhibitory deficits previously found in children with the guessing subtype of dyslexia (who read fast and inaccurately) can be attributed to dysfunctions in the fronto-central brain areas. For this purpose, the electrocortical correlates of the inhibition mechanism were assessed in a stop task that was adapted for event-related brain potential recording. It was found that in children with the spelling subtype of dyslexia (who read slowly and accurately) and normal readers, a positive component with a fronto-central scalp distribution was related to processes engaged in the inhibition of a response. Guessers did not show this “inhibition P300.” Analyses of the lateralized readiness potential (LRP) data suggested that response inhibition in spellers depended (at least in part) on their ability to inhibit the central activation of the response. In guessers, the association between response inhibition and inhibition of activity in the central motor structures was found to be weaker. It was concluded that the inhibitory deficits in guessers can be attributed to dysfunctions in the fronto-central brain structures involved in selective motor inhibition (indicated by the LRP data) and nonselective motor inhibition (indicated by the P300 data). It was suggested that there may be an association between guessers and attention deficit hyperactivity disorder children in that both clinical groups may suffer from the same type of deficits in executive functioning.

The concept of dyslexia is a heterogeneous one, and dyslexic children show a variety of disorders related to reading, such as phonological deficits, visual-orthographical deficits, and speed of processing deficits, as well as more general deficits in sequential processing, speech processing, and working memory (e.g., Castles & Coltheart, 1993; Morris et al., 1998).

To identify more homogenous groups of dyslexic children, subtypes of dyslexia have been defined (for a review, see Hooper & Willis, 1989). Although the notion of subtypes is widely accepted, the manner in which the subgroups are identified varies. For example, each of the studies listed by Hooper and Willis (1989) used different measures of achievement and cognition as the basis for group separation. In spite of this, we currently argue that the subtypes that have been distinguished by a number of the dual-subtype models—for example, Bakker's (1979, 1981) L and P types, Van der Leij's (1983) guessers and spellers type, Boder's (1970, 1973) dysphonetic and dyseidetic readers, Lovett's (1984) accuracy and rate disabled readers and Mitterer's (1982) whole-word and recoding subtypes—show some overlap and in view of their reading style seem to converge to two types of dyslexic children. The first type, henceforth referred to as guessers, manifests a fast and global reading style. This is characterized by errors such as omissions, additions, substitutions, letter reversals, false word identifications (misreading one word as another), and other word-mutilating errors. The second type, henceforth referred to as spellers, reads slowly and fragmentedly, because the identification of words is mainly based on an elaborate grapheme-to-phoneme translation process. The speller's reading style is accurate in that it leaves the ultimate reading response intact.

In this study, reading accuracy and reading speed scores on a text reading task were used to create a subtype dimension, and children with the guessing subtype of dyslexia and children with the spelling subtype of dyslexia were selected from the ends of this distribution. The children were classified as guessers and spellers according to criteria initially developed by Bakker (1981). The guesser-speller classification is based on a so-called clinical-inferential model for subtyping and has been found to cover about 60–65% of the variability in the reading of dyslexics (e.g., Van Strien, Bakker, Bouma, & Koops, 1990; Van Strien, Bouma, & Bakker, 1993). Whereas a considerable number of studies have provided validation of the guesser-speller classification (see van der Schoot, 2001, for a review), there are only a few systematic studies on its reliability. For example, Grace and Spreen (1994) reported that a subtype classification based on reading speed and reading errors is more reliable than Bakker's original method, which includes measures of dichotic listening. Clearly, more research on the reliability of the guesser-speller classification is needed.

Although empirical classification studies generally explain more variance in reading than clinical-inferential studies, they do so by extracting a larger number of subtypes (e.g., Morris et al., 1998). However, the goal of this study was not to explain as much variance in reading as possible, but to further differentiate

fast-inaccurate (guessers) and slow-accurate readers (spellers) on the basis of their brain activity during response inhibition.

Clearly, the distinction between guessers and spellers differs from the classical distinction between phonological and surface dyslexics (e.g., Castles & Coltheart, 1993; Stanovich, Siegel, & Gottardo, 1997). Whereas the former distinction refers to differences in reading style (fast-inaccurate vs. slow-accurate), the latter distinction refers to differences in deficits underlying word recognition problems (phonological vs. visuo-orthographical deficits). Yet spellers may be equated with surface dyslexics in that they are presumed to have difficulties using visuo-orthographic cues for fast, whole-word recognition (as a consequence of which they have to employ a spellinglike approach). Guessers, on the other hand, cannot be so easily equated with phonological dyslexics. Although guessers show a number of reading characteristics that are similar to the phonological dyslexia subtype, their fast, hasty reading style is hard to explain in this context.

READING AND EXECUTIVE FUNCTION

Although the differences among dyslexic subtypes generally have been described in terms of differences in deficits underlying word recognition problems, an alternative explanation stems from recent findings in the field of executive functioning (EF), thought responsible for the control of cognition, and the regulation of behavior. There is a growing body of evidence that specific patterns of executive deficits exist in childhood psychopathological disorders such as attention deficit hyperactivity disorder (ADHD), pervasive development disorder/not otherwise specified, and autism (Pennington & Ozonoff, 1996). A number of studies suggest that this may also be the case in dyslexia (e.g., Helland & Asbjørnsen, 2000; Kelly, Best, & Kirk, 1989; Purvis & Tannock, 2000). In exploring the relationship between executive dysfunctions and dyslexia more thoroughly, a number of relevant topics need to be addressed, such as comorbidity with ADHD, the domain of EF that may be affected, and the role of reading disability (RD) subtype differentiation.

Comorbidity of Reading and Attentional Disorders

The prevalence of ADHD in RD has been estimated to range from 26% to 41% (Holborow & Berry, 1986; Silver, 1981) to 50% (Lambert & Sandoval, 1980), whereas estimates of coexisting RD in ADHD range between 9% (Halperin, Gittelman, Kline, & Ruddel, 1984) and 39% (August & Garfinkel, 1990) to 80% (McGee & Share, 1988, this overview is based on Shaywitz et al., 1995). Although co-occurrence of RD and ADHD has been clearly established, the pattern of findings is not consistent, and there is yet no clear understanding of the nature of the

association. In this respect, an interesting suggestion by Cantwell and Baker (1991) is that studies need to take into account the possibility that there are subtypes of RD and ADHD, and that there is an association only among some of these subtypes.

Dyslexia and Inhibition

A number of studies using the EF approach have shown patterns of impulsivity, inattention, and distractibility in RD children that overlap with some of the criteria for ADHD, specifically, when it concerns inhibitory skills. For example, Kelly et al. (1989) found that reading disabled children have executive difficulties in selective and sustained attention, inhibition of routinized responses, and set maintenance over and above specific reading deficits. In a study by Purvis and Tannock (2000), inhibitory performance in RD and ADHD was assessed employing the classical stop task (CST; Logan & Cowan, 1984). They reported inhibitory deficits in both RD and ADHD, and they concluded that inhibitory control does not differentiate RD from ADHD. Only phonological processing was said to differentiate between these two groups. However, both studies did not specify the RD subtype.

Dyslexia Subtypes and Inhibition

Three studies assessed the extent to which subtypes of dyslexia are differentially capable of inhibiting irrelevant responses (De Sonneville, Neijens, & Licht, 1993; Licht, 1989; van der Schoot, Licht, Horsley, & Sergeant, 2000). All of these studies focused on the distinction between the slow-accurate (i.e., spelling) type and the fast-inaccurate (i.e., guessing) type.

It appeared that guessers had greater difficulty than spellers in inhibiting an experimentally induced response bias when performing a sustained attention task (De Sonneville et al., 1993), and that they were more susceptible to interference in the Stroop Color-Word Test than spellers (Licht, 1989). In a recent study by van der Schoot et al. (2000), guessers, spellers, and normal reading children were administered a classical stop task (see Logan & Cowan, 1984). It was found that guessers had inhibitory deficits, as evidenced by a lower proportion of correct inhibitions, a flatter inhibition function, and a longer stop signal reaction time (SSRT), whereas spellers were as good as controls or even better. In addition, guessers also appeared to perform more poorly than spellers on the Stroop interference task and the Tower of London planning task, suggesting that they may have more broadly distributed executive dysfunctions. Clearly, the earlier findings are in line with those reported by Purvis and Tannock (2000) in that RD children may have inhibitory deficits that are generally considered to be specific for ADHD. However, van der Schoot et al. (2000) findings suggest that these comorbid behaviors are only found in fast, inaccurate readers but not in dyslexics who read slowly but accurately.

van der Schoot et al. (2000) argued that the guessers' impulsive behaviors in both reading and executive tasks may point to a mild form of ADHD. Unfortunately, ADHD-like executive dysfunctions may go unnoticed in these children because their impulsivity does not (fully) extend to the activities to which the critical Diagnostic and Statistical Manual of Mental Disorders (4th ed. [*DSM-IV*] American Psychiatric Association, 1994) criteria for ADHD apply. Accordingly, guessers are primarily referred for their poor reading performance. Still, this does not exclude the possibility that these type of dyslexic children may have an inhibitory deficit in addition to, or underlying, their reading problem.

From a neuropsychological point of view, it would be interesting to further examine the inhibitory deficit in guessers by studying event-related brain potentials (ERPs) during inhibitory performance. There is a growing body of evidence that inhibitory deficits can be attributed to dysfunctions in the frontal and fronto-central brain areas (e.g., De Jong, Coles, Logan, & Gratton, 1990; Jodo & Kayama, 1992; Kiefer, Marzinzik, Weisbrod, Scherg, & Spitzer, 1998; Strik, Fallgatter, Brandeis, & Pascual-Marqui, 1998). In this study, guessers were compared with spellers and normal reading controls on an adapted stop task that allowed the derivation of reliable inhibition-related ERP components.

INHIBITION AND ERPS

ERPs, recorded from the scalp, represent neural activity that is time-locked to particular events (e.g., stimulus onset) in a reaction task. The ERP waveforms consist of a sequence of peaks (i.e., components) whose latencies and amplitudes have been found to vary with the speed and intensity of specific information processing stages, respectively. The registration of ERPs has several advantages over neuroimaging techniques in that it provides a real-time measure of brain activity with a high temporal resolution that enables the analysis of the chronometry of processing in the brain. In this study, we will focus on the P300 amplitude, P300 scalp topography, and lateralized readiness potential (LRP) amplitude.

P300 Amplitude

One of the most prominent ERP components that varies as a function of structural and energetic task manipulations is P300 (Sutton, Braren, & Zubin, 1965). This positive peak, occurring with a latency of 300 msec or more after stimulus presentation and a maximal amplitude over parietal scalp locations, is generally believed to be sensitive to the delivery of task-relevant information requiring a decision or response from the participant. Its latency has been found to be specifically dependent on the duration of stimulus evaluation processes, and relatively independent on the duration of response selection and execution (e.g., Kutas,

McCarthy, & Donchin, 1977; Magliero, Bashore, Coles, & Donchin, 1984). The amplitude of the P300 has typically been associated with the amount of information that is extracted from the stimulus (e.g., Ruckin & Sutton, 1978).

However, P300 amplitude is also sensitive to the requirement to inhibit a response. In a stop task, De Jong et al. (1990) found a positive deflection to be related to processes engaged in the actual inhibition of the response. This component differed from the classical P300 in that it had a fronto-central, rather than a parietal, scalp distribution. Positive components that may be equated with the "inhibition P300" of De Jong et al. (1990) have been disclosed in ERP studies by, for example, Roberts, Rau, Lutzenberger, and Birbaumer (1994), Eimer (1993), Jodo and Kayama (1992), Kok (1986), Naito and Matsumara (1994), and Podlesny, Dustman, and Shearer (1984). In this experiment, normal inhibitory processing is therefore expected to be associated with an increase in positivity at fronto-central locations.

P300 Scalp Topography

In addition to this quantitative index, we also determined qualitative P300 differences. Qualitative indexes concern the specific morphology and topographical distribution (i.e., scalp distribution) of a particular ERP component. It is assumed that qualitative ERP differences between subject groups or tasks are associated with the use of different cognitive processing modes (e.g., Courchesne, 1978).

LRP Amplitude

An ERP phenomenon deriving its validity not mainly from correlation with psychological task variables, but rather from its close relation to the response and neuroanatomy, is the LRP (see Coles, 1989; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988, for the derivation procedure). The LRP reflects differential central response activation processes in that its onset latency provides us with an unambiguous measure of the latest possible moment in time at which, in preparation of the response, one hand is being activated stronger than the other. Analogously, the amplitude of the LRP reflects the degree of selective preparation of one hand over the other.

The LRP has been used in a substantial number of studies devoted to the mechanisms and timing of motor processes (e.g., De Jong et al., 1990; De Jong, Wierda, Mulder, & Mulder, 1988; Gratton et al., 1988; Osman, Bashore, Coles, Donchin, & Meyer, 1992; Miller & Hackley, 1992). Like De Jong et al. (1990) we utilized the LRP to study the extent to which participants, when presented with a stop signal, are capable of inhibiting the central activation of a response. This ability is thought to be reflected by an LRP waveform that is "interrupted" on trials on which the response was successfully suppressed (i.e., stop signal inhibit trials, see De Jong et al., 1990).

RESEARCH OBJECTIVES AND PREDICTIONS

The goal of this investigation was to test the hypothesis that the inhibitory deficits found in children with the guessing subtype of dyslexia (van der Schoot et al., 2000; see previous discussion for a summary) can be attributed to dysfunctions in the fronto-central brain areas. For this purpose, electrocortical correlates of the inhibition mechanism were assessed in a stop task that used stop signal delays that gave a 50% chance of successful inhibition. These central delays were determined on an individual basis and were computed by subtracting the participant's SSRT from his or her primary task mean reaction time (MRT; see Method section). The SSRTs and MRTs were derived from a CST that was administered prior to the present electroencephalogram (EEG) stop task.¹ The group effects on the behavioral measures of inhibition (as observed in the CST) are reported by van der Schoot et al. (2000). In this article, we focus on the group effects on the electrophysiological measures of inhibition (as observed in the EEG stop task).

The 50% approach was considered to be advantageous for several reasons. First, indexes of inhibitory efficiency are most robust if they are determined at the 50%-inhibition stop signal delay. For example, Band, Van der Molen, & Logan (2003) found the estimation of SSRT to be most reliable around the central delay. Likewise, we expected the ERP manifestations of inhibitory processing to be most pronounced when the stop mechanism is involved in a "tight match" against the go mechanism; that is, when both have close finishing times as a result of which $P(\text{inhibit}) \cong P(\text{respond}) \cong 50\%$. Second, only the differences between waveforms for stop signal respond (SSR, i.e., failed inhibition) trials and stop signal inhibit (SSI, i.e., successful inhibition) trials are of interest for our purposes. Such differences should reflect the operation of processes that are related to the actual inhibition of the response. Conversely, ERP differences between stop signal delays are only marginally relevant and not the primary interest here. Finally, if the primary goal was to compare SSR and SSI grand average waveforms, EEG/ERP methodology requires the conditions to contain sufficient and—by preference—equal numbers of trials-to-be-averaged. Therefore, a positive side effect of the central delay approach is that 50% of the stop signal trials are available in the SSI condition and 50% of the stop signal trials are available in the SSR condition.

Compared to spellers and normal readers, we predicted that the amplitudes of the fronto-central P300 wave would be reduced in guessers. We also predicted that the central response activation processes of these children would be subjected to inhibition to a lesser extent. This would be reflected by smaller amplitudes of the LRP.

¹Because the EEG stop task directly followed the behavioral stop task, it was assumed that performance had reached a stable level and that a stop signal delay of $\text{MRT} - \text{SSRT}$ msec would yield a 50% chance of successful inhibition.

METHOD

Participants

Participants were 9- to 12-year-old children who were recruited from two special schools for children with learning disabilities and from one normal primary school. Children with learning disabilities whose reading disturbance could be attributed to emotional problems, sociocultural factors, or gross neurological deficits on the basis of school records were excluded from the sample. All children who participated ($n = 80$ for reading disabled and $n = 20$ for controls) were healthy and had normal or corrected-to-normal vision, and their IQ scores (obtained from school records) were in the normal range ($IQ > 85$). None of the children was diagnosed as ADHD using *DSM-IV* criteria, nor did they participate (or had been participating) in ADHD treatment programs.

Assessment of Dyslexia

To assess current reading level, all children were administered a standardized Dutch word-reading test (Two-Minutes Test [TMT]; Brus & Voeten, 1973), which consists of lists of words that become progressively more difficult. The TMT score, the number of words read correctly in 2 min, was converted into a reading-age equivalent (RAE; Struiksma, Van der Leij, & Veijsra, 1989) reflecting the child's actual reading level expressed in the number of months of reading instruction (1 year of instruction being equivalent to 10 months). The expected reading age (ERA) is equivalent to the number of months that a child has actually received formal reading instruction. Children who lagged 15 months or more in reading (ERA-RAE) were considered to be dyslexic ($n = 75$; 5 children with learning disabilities did not fulfill this criterion and were removed from the sample). Consequently, only those children were admitted to the subsequent guesser-speller classification procedure. All of the control children ($n = 20$) came from the normal primary school, and their RAEs approximated their ERAs.

Classification of Guessers and Spellers

Subsequent to the TMT, the dyslexic children were given a standardized Dutch sentence reading test (AVI; Van den Berg & Te Lintelo, 1977). This test consists of nine texts with increasing difficulty. The number of texts actually mastered (i.e., read within time and error limits) determines the child's mastery level of text reading.

The AVI was employed to classify the dyslexics as spellers or guessers on the basis of reading speed, the number of substantive errors (SE; e.g.,

omissions, additions, substitutions, and letter reversals) and the number of time-consuming errors (TE; e.g., hesitations, stammerings, fragmentations, repetitions, and corrections). To evoke a sufficient number of errors on which to base the speller (relatively many TEs)–guesser (relatively many SEs) classification, a text that was two levels above the child's mastery level was presented and assessed on reading speed (RS) and reading errors.

RS was expressed as the total reading time divided by the time norm for the text $\times 100$, whereas reading error (RE) was expressed as the proportion of TE errors relative to the total number of errors (SE + TE). A child was classified as the guessing type of dyslexia when RS was less than 115 and RE was less than .40 (more than 60% of errors made were SEs), and as spelling type of dyslexia when RS was greater than 135 and RE was greater than .60 (more than 60% of errors made were TEs). The classification criteria were similar to those used earlier (e.g., Van Strien, 1999) and adapted from Bakker and Vinke (1985). Using this classification system, we were able to classify about 60% of the dyslexics as either spellers or guessers ($n = 45$). The final groups of guessers ($n = 20$) and spellers ($n = 20$) were formed by selecting those children who showed most clearly the characteristics of each type.

Symptoms of ADHD in Dyslexics

To evaluate possible comorbid ADHD symptoms in the sample of dyslexics, teachers rated the dyslexic children and controls with the Abbreviated Conners Teacher Rating Scale (ACTRS; Goyette, Conners, & Ulrich, 1978). A one-way analysis of variance (ANOVA) performed on the rating scores revealed a significant group effect, $F(2, 56) = 9.51, p < .001, \eta = .254$. As expected, guessers displayed higher scores than controls ($p < .001$) and spellers ($p < .08$). Group characteristics are presented in Table 1.

STOP TASK AND PROCEDURE

In the EEG stop task, participants were asked to respond to a visual stimulus and suppress their response in the unlikely event of a stop signal (Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984). For each participant, stop signals were exclusively presented at the most central delay, where the probability of inhibition is 50%.

Setting the 50% Inhibition Delay

To obtain the 50% inhibition delay, SSRT was subtracted from the participants MRT on the primary task. It was important that both parameters could be

TABLE 1
Characteristics for Each Reading Group

Reading and Spelling Achievement																		
			Reading Age ^a										Reading Speed ^b (on AVI)		Error Type ^c (on AVI)		Conners Rating Scale	
			Age		Expected		Actual		Difference									
			Boys	Girls	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
	Spellers	13	7	10.5	1.0	41.7	11.4	21.1	10.1	20.6	5.0	153.9	29.8	.67	.14	6.5	6.8	
Guessers	14	6	10.6	1.0	40.4	10.2	18.2	6.6	22.2	6.9	84.7	21.4	.30	.12	10.8	8.0		
Controls	12	8	10.9	0.4	40.0	5.6	42.1	7.1	-2.1	9.3	—	—	—	—	2.1	2.1		

^aReading age is in months. Ten months equals 1 year of reading instruction. (The actual reading age is derived from the Two-Minutes-Test [Brus & Voeten, 1973]). ^bReading speed is expressed as $100 \times (\text{time needed}/\text{time norm})$. ^cError type is expressed as $N(\text{time-consuming errors})/N(\text{substantive} + \text{time-consuming errors})$.

derived—for each individual—from a CST (Logan & Cowan, 1984) that preceded the present EEG stop task. As noted previously, the behavioral measures of inhibition that stem from the CST are reported elsewhere (van der Schoot et al., 2000). As illustrated in Figure 1, a stop signal presented MRT–SSRT msec after the onset of the go signal causes the stop process and the go process to have equal finishing times.

Specifically, the 50% inhibition delay divides the primary task reaction time distribution into two halves. The left half corresponds to the probability of response (50%) and consists of the responses fast enough to escape inhibition; the right half corresponds to the probability of inhibition (50%) and consists of the trials on which the stop process beats the go process.

It should be emphasized that the above logic is only legitimate under the assumptions of the race model: (a) Primary task processes and stop processes proceed independently; (b) their finishing times vary randomly over trials; and (c) SSRT is a constant. In addition, it was assumed that performance on the primary-choice reaction time task had reached a stable level and would continue to vary around the MRT that was found in the CST.

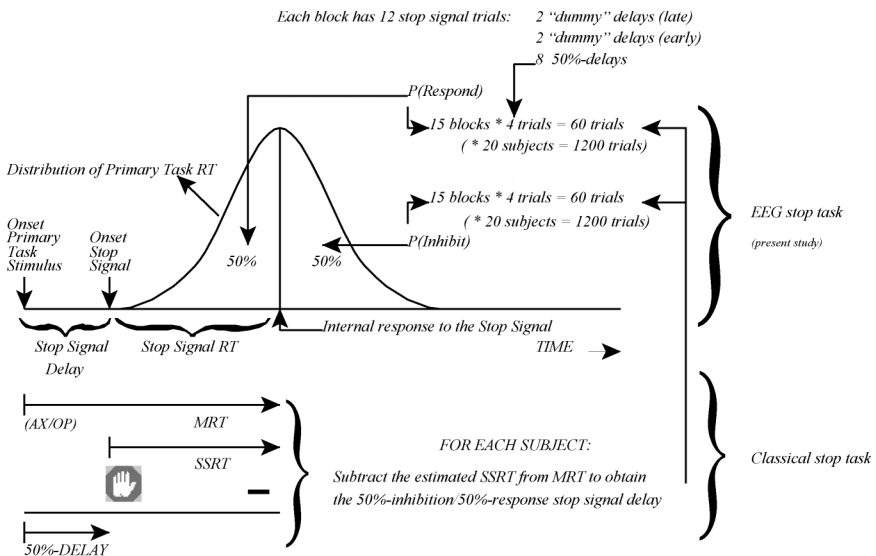


FIGURE 1 Graphical representation of the assumptions and predictions of the race model, indicating how the mean reaction time–stop signal reaction time (MRT–SSRT) stop signal delay in the electroencephalogram (EEG) stop task is expected to yield 50% stop signal inhibit and 50% stop signal respond trials. To establish the participants MRT and SSRT, the EEG stop task was preceded by a classical stop task (see text). (AX/OP) reflects the onset of the go signal; the stop sign reflects the onset of the stop signal.

Setting Additional “Dummy” Delays

As Logan (1994) advocated, a single-delay method endangers the validity of the stop task. That is, if there is only one stop signal delay, participants learn to appraise the moment in time at which the stop signal is presented. Consequently, they may prolong their responses to the primary task—awaiting the possible occurrence of the stop signal—to enhance their probability of inhibiting. However, this strategy can be minimized by “presenting several delays, some early enough that participants will be able to inhibit most of the time, and some late enough that participants will usually respond when they occur” (Logan, 1981). Therefore, we decided to employ early and late delays in addition to the 50%-inhibition delay. These “dummy” delays were not included in the ERP analyses; their only purpose was to meet the requirements of the stop task procedure.

Primary Task

Each trial began with the presentation of a square warning stimulus (1.40 cm × 1.40 cm) illuminated for 500 msec. It was followed by the primary task stimulus, which was displayed for 125 msec. After the imperative signal was extinguished, the screen went blank for a intertrial interval of 2,375 msec. The stimuli for the primary task were the uppercase letters *X*, *A*, *O*, and *P*. Each letter was 1.80 cm wide and 2.90 cm high. Both the warning stimuli and the stimulus letters were presented in black-on-white and in the center of the screen. The primary choice reaction time task was the same as in the CST: A capital *X* or *A* required a response with one hand; a capital *O* or *P* required a response with the other. Mapping of letters onto the response box was counterbalanced across participants.

Stop Signals

A stop signal was presented on 25% of the trials, occurring equally often with an *X*, *A*, *O*, and *P*. The sequence of primary task stimuli, stop signals, and stop signal delays was pseudorandomized. The stop signal was a 1000-Hz tone, with an intensity of 65 dB(A) and duration of 350 msec. It was presented binaurally by size-adjustable, padded headphones.

A total of 15 blocks of 48 trials were administered. In each block, eight stop signals were presented at the 50%-inhibition delay, and four stop signals were presented at a dummy delay (two early delays + two late delays). Consequently, about 60, $(15 \times 8)/2$, trials were expected to end up in the stop signal inhibit condition, and 60 trials were expected to end up in the stop signal respond condition (see Figure 1).

Instructions

Instructions for the primary-choice reaction time task were given first. Participants were told to respond as fast and accurately as possible. Then the participants were instructed to try to withhold the response whenever a stop signal occurred. It was clarified that stop signal delays were varied by the experimenter in such a way that sometimes stop signals would be presented so late that it would be extremely difficult to suppress the primary response. Finally, participants were explicitly instructed not to delay their responses to the go task to improve the odds of stopping.

Task Duration

In total, the EEG stop task lasted about 1 hr. The test blocks were arranged in groups of three. A short break was scheduled after each part. The CST that preceded the present stop task (previously discussed) lasted about half an hour. In between the two tasks, the participants took a rest of at least 45 min, during which the electrocap was attached and the 50% inhibition delay was determined. Meanwhile, the participants watched a movie.

APPARATUS

Stimuli were presented with a 386SX-25 PC, with timing control from a master computer, a 486DX2-66 PC. The master computer recorded the manual and electrophysiological responses. The stimuli were presented on a NEC Multisync 5FG monitor positioned at 70.00 cm from the participants eyes. Participants lay on a bed in a dimly illuminated and electrically shielded cubicle. On either side of the bed, a response box was positioned at an optimal location for each participant.

ELECTROPHYSIOLOGICAL RECORDING

The EEG was recorded from FP1, PFz, FP2, F7, F3, Fz, F4, F8, FC3, FCz, FC4, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, Oz, and O2, with linked earlobes as reference. The electrodes were mounted in an electro-cap (Electro Cap International) and arranged according to the 10/20 system (Jasper, 1958). Bipolar recordings of the horizontal and vertical electrooculogram (EOG) were made with tin electrodes placed at the outer canthi of both eyes, and at infraorbital and supraorbital locations in line with the pupil of the right eye, respectively. A ground electrode was positioned at AFz. Electrode impedance was kept below 5 k Ω for both the EEG and EOG electrodes. The EEG/EOG signals were amplified with a high frequency cutoff at 35 Hz and a

time constant of 5 sec for the high-pass filter. For each measure (EEG and EOG) on each trial, the derived voltages were digitized at 100 Hz for 2,560 msec, starting 100 msec before the onset of the warning stimulus.

DATA ANALYSIS

Behavioral Data

For each participant, the primary task MRT and the standard deviation of the MRT were derived from the go trials. In addition, the SSRT was estimated, taking into account the probability of response that was actually observed at the 50% delay and the distribution of the primary-task reaction times (see Logan & Cowan, 1984).

Electrophysiological Data

Trials with A/D converter saturation, movement artifacts (difference among consecutive time points exceeding $\pm 100 \mu\text{V}$), flat-line recordings (longer than 50 msec) and amplitude drifts (difference between maximum and minimum amplitude within a "sweep" of 256 samples exceeding $200 \mu\text{V}$) were excluded from further analysis. Occular artifact in the EEG was estimated and corrected for by regression analysis in the frequency domain (Woestenburg, Verbaten, & Slangen, 1983).

For each channel, average stop-stimulus-locked ERPs were computed separately for SSIs (successful inhibition), SSRs (unsuccessful inhibition), and for the no-stop signal trials from the parts of the primary task reaction time distribution that correspond to the proportions of SSI and SSR trials. The latter were classified with respect to whether RT was longer (no-stop signal inhibit [nSSI]) or shorter (no-stop signal respond [nSSR]) than the finish time of the stop process (i.e., 50% inhibition delay + SSRT). Subsequently, the ERP for the nSSI trials was subtracted from the ERP for the SSI trials, and the ERP for the nSSR trials was subtracted from the one for the SSR trials. Because all brain activity related to the processing of the go signal (the *A*, *X*, *O*, or *P*) is subtracted out, these differences should presumably reflect the processing of the stop signal (in the SSI–nSSI and the SSR–nSSR condition), and, more importantly, the successful operation of subsequent inhibitory processes (in the SSI–nSSI condition).

The resulting difference waves were obtained over an interval of 800 msec, starting 100 msec before the time of onset of the stop signal. They were low-pass filtered (i.e., smoothed) with a two-step (–3dB at 6.0 Hz) moving average filter (Ruckin & Glaser, 1978). Finally, the mean amplitude of the 100 msec prestimulus baseline period was subtracted from the time series.

P300 amplitude. P300 amplitude was scored by means of a peak picking procedure for each participant and condition (SSI and SSR). It was defined as the maximum amplitude in the average waveform recorded from the central (Cz) and fronto-central (FCz) electrodes between 150 and 450 msec poststimulus. The electrode locations and time window were determined after visual inspection of the individual participant and grand average ERPs.

P300 amplitudes were submitted as dependent variables to an ANOVA for repeated measurements. The within-subject factor was Inhibition (with two levels: SSI and SSR) and the between-subject factor was Group (with three levels: guessers, spellers, and controls). When necessary, follow-up tests were carried out to locate between-group differences.

P300 scalp topography. To assess group differences in the scalp topography of P300, amplitudes were derived from frontopolar (FP1, PFz, FP2), frontal (F7, F3, Fz, F4, F8), fronto-central (FC3, FCz, FC4), central-temporal (T3, C3, Cz, C4, T4), parietal-temporal (T5, P3, Pz, P4, T6), and occipital (O1, Oz, O2) scalp locations. For each participant, they were obtained at the peak-latency identified at Cz, where the peak was most pronounced. The data were subjected to ANOVA with one between-subject factor (i.e., Group, 3 levels: guessers, spellers, and controls) and repeated factors of Inhibition (2 levels: SSI and SSR) and Electrode (24 levels: FP1, FPz, ..., Oz, O2). The multivariate approach (MANOVA) was used to avoid problems concerning sphericity (O'Brien & Kaiser, 1985; Vasey & Thayer, 1987).

Because of the incompatibility between the additive model underlying ANOVA and the multiplicative effects on ERP's voltages produced by differences in source strength (McCarthy & Wood, 1985), differences in overall amplitude may masquerade as changes in scalp distribution. The data were therefore normalized prior to the MANOVA. For each condition and participant group, the P300 amplitudes at a particular electrode site were converted to a percentage of the range between the minimum amplitude and maximum amplitude across all electrodes. This normalization procedure allowed for an examination of qualitative scalp distribution differences unaffected by absolute differences in amplitude between groups or conditions (McCarthy & Wood, 1985). Differences in topographical distribution would be evidenced by interactions between the Electrode factor and Group and Inhibition. If significant, additional follow-up analyses (simple effects; Winer, 1971) were conducted to clarify the interactions.

LRP amplitude. The LRP was computed from the noncontaminated trials (previously described) by means of the following two-step procedure (Coles, 1989). First, the average C3–C4 difference potentials were calculated separately for left- and right-hand responses. Then, the (C3–C4) time series for left-hand responses and the (C3–C4) time series for right-hand responses were averaged.

The LRPs were derived separately for SSI, SSR, nSSI, and nSSR trials (previously discussed). They were computed over an interval of 1,800 msec, starting 100 msec before the time of onset of the go signal. The 100-msec prestimulus period was used as a baseline for amplitude measurements. To capture the most interesting lateralization that is associated with differential response preparation, the LRP averages were subjected to a filter bandpass of 2.0 to 6.8 Hz (with 3 dB/octave). LRP amplitude was scored by means of a peak picking procedure for each participant and condition. It was defined as the maximum amplitude in the average waveform between 500 and 1,100 msec poststimulus.

In the LRP analysis, the maximum LRP amplitudes were compared by means of an ANOVA with one between-subject factor (Group; three levels: guessers, spellers, and controls) and repeated measures across Inhibition (four levels: SSI, SSR, nSSI, and nSSR).

RESULTS

Behavioral Data

One-way ANOVAs were conducted separately for the MRT, the standard derivation of the MRT, and the SSRT. Means and standard deviations of each of these dependent measures in each participant group (spellers, guessers, and controls) are presented in Table 2.

Significant group effects were obtained for the MRT, $F(2, 57) = 8.62, p < .001$ (spellers and guessers had longer RTs than normal readers) and the standard derivation of the MRT, $F(2, 57) = 13.77; p < .000$ (spellers and guessers showed a greater amount of variability than normal readers). The group effect on SSRT was marginally significant, $F(2, 57) = 2.46, .05 < p < .1$ (guessers had a slower stopping process than spellers and controls).

TABLE 2
Performance on the Stop Signal Paradigm as Reflected by the Means and Standard Deviations for the Dependent Measures for Each Reading Group

Measure	Reading Group					
	Spellers		Guessers		Controls	
	M	SD	M	SD	M	SD
MRT (go task)	807.67	112.26	807.83	103.94	692.15	87.13
SD of the MRT	345.13	78.28	343.77	97.43	225.04	71.40
SSRT	208.58	166.69	324.98	242.27	215.60	132.22

Note. MRT = mean reaction time; SSRT = stop signal reaction time. All times are in ms.

Electrophysiological Data

It should be recalled that we predicted 50% of the stop signal trials to fall in the SSI condition and 50% of the trials to fall in SSR condition. This assumption proved to be warranted only in guessers. In controls and spellers, the ratios were found to be 53–47% and 45–55%, respectively.

P300 amplitude. The grand average stop stimulus-locked ERPs at Fz, FCz, Cz, Pz, and Oz for both of the inhibition levels (SSI–nSSI and SSR–nSSR) are displayed in Figure 2 for controls, spellers, and guessers separately. The waveforms appear to consist of an early negative deflection that peaked around 125 msec and a later positive deflection that was largest in amplitude approximately 300 msec following the onset of the stop signal. The former corresponds to the N1 component,

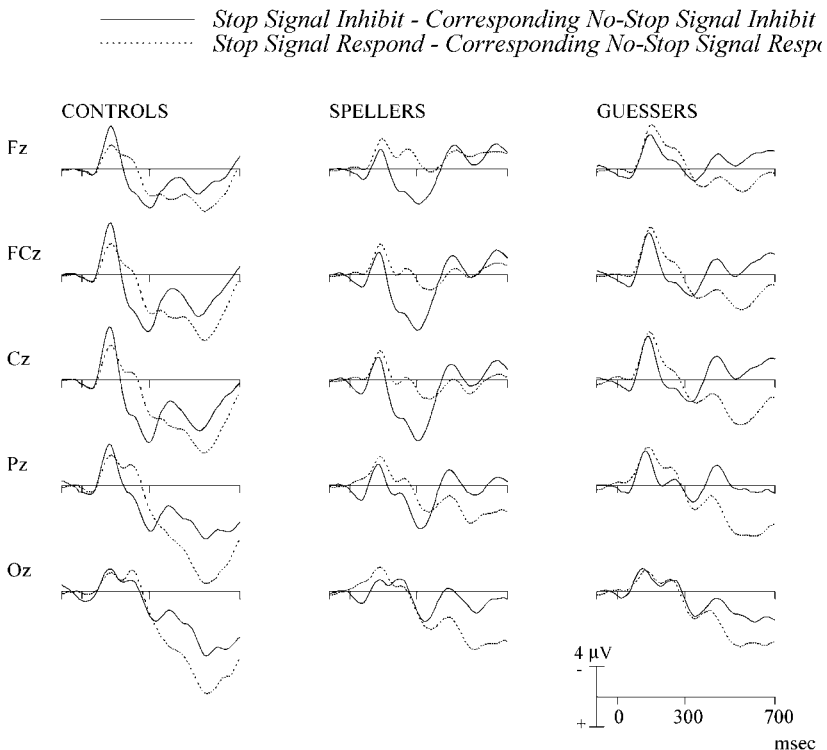


FIGURE 2 Grand average stop stimulus-locked waveforms for controls (left column), spellers (middle column), and guessers (right column) at Fz, FCz, Cz, Pz, and Oz. The waveforms represent the difference between the stop signal inhibit and stop signal respond trials, and the corresponding no-stop signal trials.

which has been found to be evoked by the presentation of auditory stimuli (Näätänen & Picton, 1987). The latter may be equated with the inhibition P300 (e.g., De Jong et al., 1990; Eimer, 1993; Jodo & Kayama, 1992; Kok, 1986; Naito & Matsumara, 1994; Podlesny et al., 1984). As can be seen in Figure 2, P300 was more positive for stop signal inhibit trials than for stop signal respond trials, especially at Cz and FCz. More important, the difference in positivity was larger for spellers and (to a lesser extent) controls than for guessers. Two types of statistical analyses were performed on the data: (a) traditional analyses of P300 amplitude at the electrode locations where P300 is most predominant (i.e., Cz and FCz) and (b) analyses of P300 scalp topography.

The ANOVA on the P300 peak amplitudes in the 150- to 450-msec range yielded a significant main effect for Inhibition at Cz, $F(1, 57) = 19.67, p < .001$, and FCz, $F(1, 57) = 16.94, p < .001$. Tests for the inhibition effect for each group revealed a significant difference for spellers (Cz and FCz: $p < .001$) and controls (Cz and FCz: $p < .05$). The Group \times Inhibition interaction effect was marginally significant: Cz: $F(2, 57) = 2.74, p < .08$; FCz: $F(2, 57) = 3.07, p < .06$. Simple effect tests subsequently demonstrated the interaction to be significant in the speller–guesser comparison (Cz and FCz: $p < .05$).

A striking aspect of the ERP data is that in the SSI condition only the controls showed an enhancement of the negative-going waveform (N1) that preceded the P300. Consequently, this may have affected the baseline from which the P300 developed. In order to account for this possibility, we decided to redefine P300 amplitude as the peak-to-peak difference in a 50- to 425-msec time window. This scoring procedure resulted in a significant Group \times Inhibition interaction, Cz: $F(2, 57) = 3.68, p < .05$; FCz: $F(2, 57) = 3.63, p < .05$. The interaction was also significant in the (post hoc) control–guesser comparison (Cz and FCz: $p < .01$).

In addition to the effects on the P300, Figure 2 shows that Group and Inhibition (SSI vs. SSR) had an unanticipated effect on the positivity in the 300- to 700-msec time period. From Figure 2, it can be seen that the P550 was larger in controls than in spellers and guessers and that failed inhibition (SSR) trials evoked a larger P550 than successful inhibition (SSI) trials. These effects were largest at Pz and reached conventional levels of significance in a post hoc analysis, Group: $F(1, 57) = 14.53, p < .001$; Inhibition: $F(2, 57) = 6.63, p < .005$. Although Figure 2 suggests that the Inhibition effect on P550 was more manifest in controls and guessers than in spellers, the Group \times Inhibition interaction was not significant, $F(2, 57) = 1.59$.

P300 scalp topography. A repeated-measures MANOVA (using the multivariate test statistic Wilks's lambda) on the normalized P300 amplitudes derived from 24 electrodes across the scalp yielded a significant Inhibition \times Electrode interaction, $F = 3, 24, p < .01$. The interacting effects of Inhibition and Electrode tended to vary as a function of Group, as was evident in the second-order interaction Group \times Inhibit \times Electrode, $F = 1.45, p < .08$.

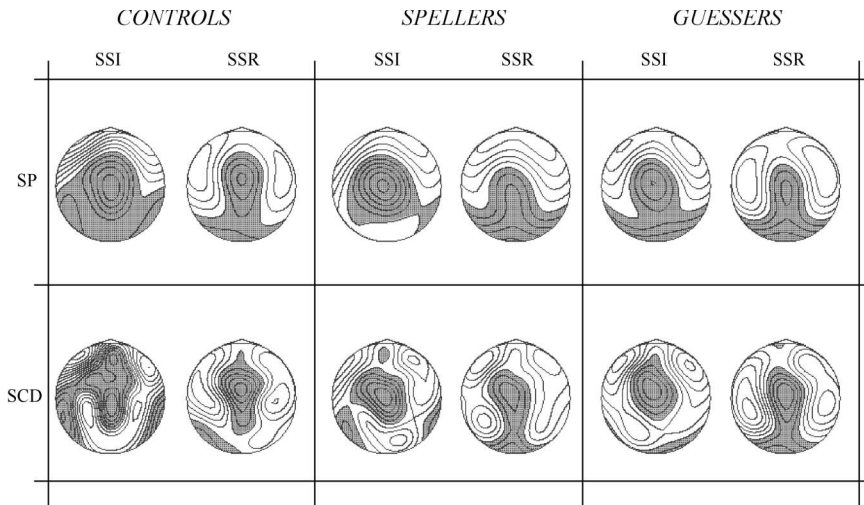


FIGURE 3 Scalp potential (SP) and scalp current density (SCD) isocontour maps for SSI and SSR trials. They were computed at the P300 peak latency of the grand average Cz-waveform for controls (296 msec, poststimulus), spellers (306 msec), and guessers (326 msec). The isocontour lines for the SP maps are separated by $0.8 \mu\text{V}$; for the SCD maps, they are separated by $0.4 \mu\text{V}/\text{cm}^2$. Gray areas indicate positive voltages or outward current flow; white areas indicate negative voltages or inward current flow.

The statistical results were confirmed by the scalp potential (SP) isocontour maps (presented in Figure 3, upper row), obtained at the P300 peak latency of the grand average Cz waveform for controls (296 msec, poststimulus), spellers (306 msec), and guessers (326 msec). Although they were constructed at a single time point, the topographic features of the maps were constant over a fairly large time window centered around each of these latencies.

In spellers and controls, the P300 scalp topographies varied as a function of Inhibition. In the SSI condition, they were characterized by a clear fronto-central focus. In the SSR condition, the topographic profiles were far less distinct. That is, the substantial decrease in amplitudes was paralleled by a slight shift toward a more centro-parietal P300 distribution (in spellers), or by a “narrowing” of the central positivity (in spellers and controls). Follow-up tests demonstrated that the Inhibition \times Electrode interaction was significant in both groups ($p < .05$). Visual inspection of the scalp potential maps of guessers indicated that there were no substantial differences in P300 scalp topography between the SSI and SSR conditions.

In the SSI condition, the scalp current density (SCD) maps associated with the SP maps (see Figure 3, lower row) indicated the presence of outward current maxima (i.e., current sources) at the fronto-central scalp locations, and inward current maxima (i.e., current sinks) at the lateral frontal locations. In the SSR condition, the outward currents were weaker and showed a more central focus. These effects were

most evident in spellers and controls. The pattern of inward current flow was more variable across groups. In controls, the bilateral current sinks changed from a frontal location to a central location. In guessers, they shifted to more centro-parietal regions. In spellers, the SCD maps disclosed a distribution of inward current maxima near lateral frontal and lateral centro-parietal scalp locations. In all groups and conditions, stronger currents were found over the left than over the right hemisphere.

LRP amplitude. LRPs were computed for SSI trials, SSR trials, and for the no-stop signal trials from the parts of the primary task reaction time distribution that correspond to the proportions of SSI and SSR trials. The LRPs were very noisy. Four children (2 controls, 2 spellers) did not even display any positivity between 300 and 900 msec poststimulus. These children were excluded from further analyses. The grand average LRP waveforms are depicted in Figure 4. A repeated-measures ANOVA on the maximum amplitudes yielded a significant main effect of Inhibition, $F(3, 159) = 3.68, p < .05$. The amplitude effects were similar for each of the reading groups, as was reflected by the nonsignificant Group \times Inhibition interaction, $F(6, 159) = .95$. As these results clearly need some clarification, we will elaborate on several of the most striking aspects of the LRP waveforms.

As can be seen from Figure 4, the LRP waveforms for SSR trials and the corresponding nSSR trials largely overlap in controls and guessers. This suggests that central response activation processes remain unaffected by the processing of the stop signal on SSR trials. It is important that this result validates the assumption of the race model that primary-task processing and stop signal processing are independent. Although the LRP data of the spellers seem to contradict the hypothesized SSR/nSSR overlap, an ANOVA confirmed that the waveforms—pooled across reading groups—reached similar levels of activity in both conditions, $F(1, 53) = .61, ns$.

Another main feature of Figure 4 is that in all groups the LRP in the nSSI condition was much weaker than the LRP in the nSSR condition. Because these waveforms reflect response activation processes on trials from, respectively, the slow and fast part of the primary-task reaction time distribution (see Figure 1), we anticipated them to differ in onset latency but not in amplitude. Two explanations may account for this finding.

First, the flattening of amplitude may be an artifact of having averaged over trials across which the RTs—and thus the LRP onset latencies—varied substantially (i.e., “latency jitter”). As RT distributions are typically skewed to the right, the trials of which the nSSI and nSSR waveforms are composed may have come from subdistributions that not only differed in mean but also in variability. To explore this possibility in controls, we split the primary-task RT distribution of the controls into the nSSI distribution and nSSR distribution. The resulting “slow bin” and “fast bin” distribution are shown in Figure 5. As predicted, the former appeared to be flatter than the latter. More important, by comparing the distributions with the corresponding nSSI- and nSSR-LRPs, we were able to assess the confounding effects of increased variance across RT (i.e., LRP onset latencies) on the grand average amplitudes.

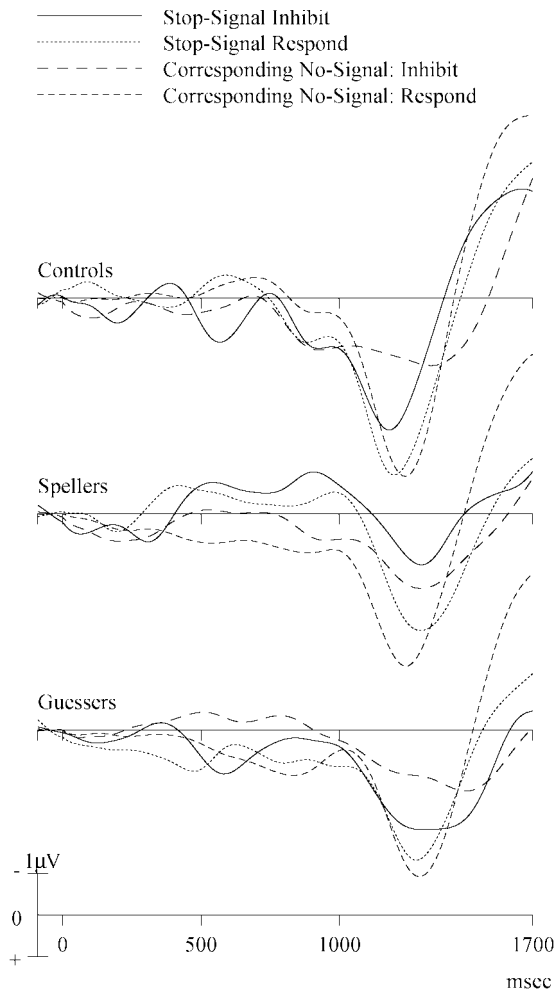


FIGURE 4 Waveforms of the lateralized readiness potential for controls, spellers and guessers for stop signal inhibit trials, stop signal respond trials, and the corresponding no-stop signal trials. The warning signal was presented at $t = 0$ msec, the go signal was presented at $t = 500$ msec.

Second, the decrement in amplitudes in the nSSI condition may have been due to an overlap of trials on which the LRP displays a correct (positive) lateralization and trials on which the LRP displays an incorrect (negative) lateralization prior to going in the correct direction. The latter type of trials (i.e., trials on which there is an initial activation of the incorrect hand) prevails at the slow bin distribution (see Smulders, Kenemans, & Kok, 1996). Because the incorrect lateralization trials overlap the correct lateralization trials, the averaging

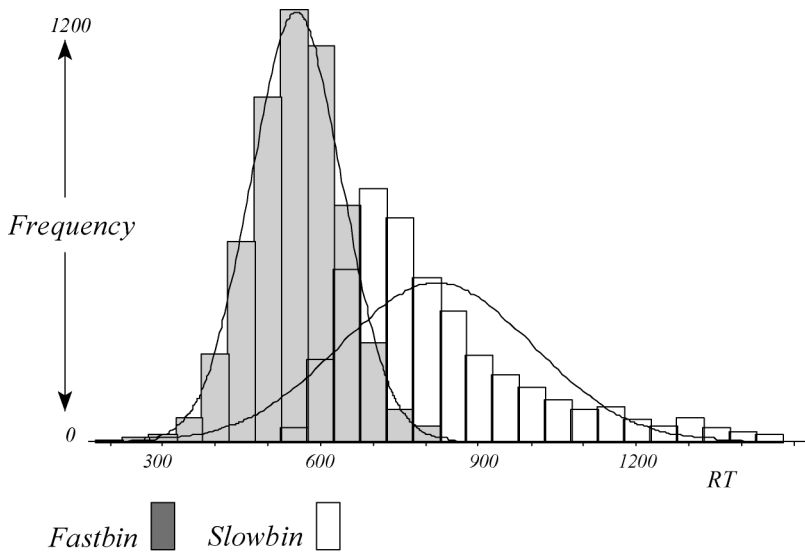


FIGURE 5 The primary task reaction time distribution of the controls split into the no-stop signal inhibit distribution (“slow bin”) and the no-stop signal respond distribution (“fast bin”).

procedure is expected to flatten the resulting LRP amplitudes, especially in the nSSI condition.

The most important aspect of the LRP data concerns the maximum amplitudes of the SSI waveforms. While inspecting and analyzing them, we ignored the “deviant” nSSI-LRPs, as they would only obscure the analysis (previously described).

Although initially the SSI-LRPs appeared to develop normally, they were subsequently interrupted in each of the participant groups. This effect was most pronounced for spellers. In guessers, the SSI waveform reached an amplitude that approximated the nSSR/SSR maximum. This pattern of results was only partly confirmed by the statistical analyses. An ANOVA that tested the special contrast $\text{SSI} - (\text{SSR} + \text{nSSR})/2$ failed to show a significant $\text{Group} \times \text{Inhibition}$ interaction, $F(2, 53) = 1.35$, *ns*. Additional tests for each group indicated that the difference was significant only in spellers ($p < .05$).

Finally, inspection of Figure 4 indicates that in controls the SSI-LRP seems to peak earlier than the SSR-LRP and nSSR-LRP, and that in spellers and guessers, the SSI-LRP seems to peak later than the SSR-LRP and nSSR-LRP. However, an ANOVA on the LRP peak latencies that tested the $\text{SSI} - (\text{SSR} + \text{nSSR})/2$ contrast failed to show a significant $\text{Group} \times \text{Inhibition}$ interaction, $F(2, 53) = .15$.

Probably, the visual features of the grand average LRPs could not be fully retained in the analyses because the single-participant LRPs were noisy and displayed a great amount of between-subject variability.

DISCUSSION

The main goal of this study was to examine the electrophysiological correlates of the inhibition mechanism in dyslexic children. Implicitly, we wanted to test the hypothesis that the inhibitory deficits that were previously found in children with the guessing subtype of dyslexia can be attributed to dysfunctions in the fronto-central brain areas. This hypothesis was first put forward by van der Schoot et al. (2000). These authors found lower and flatter inhibition functions and longer SSRTs for guessers than for spellers when employing a CST. In addition, guessers performed more poorly on the Stroop interference task and the Tower of London task, and they displayed higher rating scores on the ACTRS. These findings led the authors to conclude that guessers may have a broader underlying executive deficit, which in turn may be attributable to dysfunctions in the fronto-central brain areas.

Inhibition Deficits in Guessers: Behavioral Evidence

Clearly, the predicted guesser–speller differences were also evident in the performance on the stop task adapted for ERP recording in this study. In comparison with spellers, guessers were found to have a slower inhibitory process, as reflected in a longer SSRT (325 msec vs. 209 msec). This finding corroborates that reported by van der Schoot et al. (2000) and confirms that guessers are impaired in their ability to inhibit responses that have become inappropriate.

A number of studies support the idea that dyslexic children have executive deficits. These deficits were reflected by poor response inhibition (Purvis & Tannock, 2000), poor flexibility of responding (Helland & Asbjørnsen, 2000), increased Stroop interference (Evarett, Warner, Miles, & Thomsen, 1997), planning and organizational problems (Condor, Anderson, & Saling, 1995; Levin, 1990) and difficulties in selective and sustained attention (Kelly et al., 1989). Only the Helland and Asbjørnsen (2000) study distinguished among subgroups of dyslexics, showing that dyslexic children with more pronounced receptive language impairments experienced more executive deficits.

The Inhibitory Deficit in Guessers: A Central or Peripheral Deficit?

The electrophysiological data recorded during performance on the stop task were in line with the behavioral data. It should be recalled that stop signals were exclusively presented at the participants' most central delay, where the probability of inhibition is 50% and the ERP manifestations of inhibitory processing are

thought to be most pronounced. Of special interest was the amplitude of P300 and the LRP.

In controls and spellers, the amplitudes of P300 appeared to be related to processes that differentiated between stop signal inhibit and stop signal respond trials, that is, to those processes related to the actual inhibition of the response. In addition, the scalp topography of P300 varied as a function of the effectiveness of response inhibition. In the case of successful inhibition, it had a fronto-central focus. In the case of failures of response inhibition, the potential maps demonstrated a narrower distributed positivity at more centro-parietal scalp areas. Guessers showed neither quantitative P300 differences (amplitudes) nor qualitative P300 differences (scalp topography) between the stop signal inhibit and the stop signal respond condition. Their ERPs were characterized by an absence of inhibition-related P300 activity.

An increasing number of ERP studies report that fronto-central brain structures are involved in response inhibition (Fallgatter & Strik, 1999; Strik et al., 1998). Although in several studies, response inhibition was associated with enhanced frontal N2 amplitudes, other studies revealed enhanced P300 components at fronto-central locations when responses had to be inhibited (e.g., Eimer, 1993; Fox, Michie, Wynne, & Maybery, 2000; Roberts et al., 1994). The involvement of fronto-central brain structures in response inhibition has been confirmed by topographical and source localization analyses of the inhibition-related ERPs (Fallgatter, Brandeis, & Strik, 1997; Fallgatter & Strik, 1999; Strik et al., 1998). These analyses revealed a clear anteriorization of P300 in a no-go inhibition condition relative to a go respond condition.

Analyses of the LRP data suggested that response inhibition in spellers depends (at least in part) on their ability to inhibit the central activation of the response. This ability was evident in the interruption of the LRP waveform on trials on which the response was successfully inhibited. In guessers, the association between response inhibition and inhibition of activity in the central motor structures was found to be weaker. This result matches with the pattern of results found for P300.

It has been suggested that the fronto-central P300 component reflects the operation of a peripheral inhibition mechanism that is hypothesized to function concurrently with a central mechanism whose inhibitory actions are reflected by the LRP (De Jong et al., 1990). Whereas the central mechanism is charged with preventing the outflow of central motor commands, the peripheral mechanism aims (if necessary) at the interception of such outflow as it is being transmitted to the peripheral motor structures. It is important that the latter mechanism can only be applied when ongoing actions have to be interrupted nonselectively (as in this experiment). For a more extensive discussion on the distinction between both inhibitory mechanisms, one is referred to De Jong et al. (1990).

Interpreting the Group Differences in the Light of the 50% Approach

At first glance, the LRP/P300 differences among the dyslexia subgroups seem to provide evidence in support of the hypothesis that the inhibitory deficits in guessers can be attributed to dysfunctions in the fronto-central brain structures involved in selective motor inhibition (indicated by the LRP data) and nonselective motor inhibition (indicated by the P300 data). However, a more careful examination of the between-group differences necessitates their interpretation in the light of the applied 50% method. In this stop task, stop signals were presented MRT-SSRT msec after the onset of the go signal. This procedure guaranteed a tight match between the go process and the stop process on the majority of trials (see Figure 1). At the same time, it reduced the number of trials at which a stop signal is presented too late (in that inhibition can only be successful thanks to disturbances in the primary-response process) or too early (in that inhibition could succeed without effort, as primary-response activation has not even been started up yet). Thus, on only a few of the stop signal inhibit trials, the ERP signature might have reflected spurious inhibition-related processes. On the majority of them, the stop mechanism was tapped while it was in "full operation."

However, a possible disadvantage of the 50% approach is that the central stop signal might have "hit" different stages of response processing in children with a slow inhibitory process (i.e., guessers) and children with a fast inhibitory process (i.e., spellers). Given the fact that the primary response times in the EEG stop task were identical for guessers and spellers, this means that stop signals intersected the reaction process earlier in guessers than in spellers. Of concern is the incidence of trials on which the stop signals hit the sequence of response processing stages in guessers too early, that is, on which inhibition could succeed without effort because primary response activation was only just commenced or not yet started up. A high proportion of this type of stop trial would explain the finding that overt response inhibition in guessers—as observed on 50% of the stop signal trials—is not manifest in their LRP and P300. Yet the observation that guessers did not succeed in suppressing the response on the other 50% of the trials weakens an interpretation in terms of lower stop task demands. In fact, it implies that guessers had good reason to bring the stop mechanism into action but, unfortunately, were not able to do so effectively. As demonstrated by the van der Schoot et al. (2000) study, the incapacity to (fully) deploy the stop mechanism is detrimental to the ability to inhibit inappropriate responding throughout a regular stop task, that is, a stop task that does not "spare" those who have a slow SSRT.

This leaves us with the question of how to explain the dissociation between the behavioral and electrocortical findings in guessers. Guessers were able to

inhibit their responses on 50% of the trials, but there was no inhibition-related P300 augmentation or LRP interruption similar to that found in controls and spellers. Figure 2 shows that stop signals did elicit a N1 response in guessers, indicating that the stop signal was detected and processed. Therefore, it seems necessary to assume that in guessers, inhibitory processes other than those reflected by the P300 and LRP were responsible for the inhibition of the responses. Still, we argue that, regardless of whether this assumption is valid or not, the outcomes of this study suggest that the inhibition problem in guessers is linked with dysfunctions in the motor-related fronto-central cortical areas.

Finally, the finding that failed inhibition trials evoked a larger P550 than successful inhibition trials needs to be discussed. This effect was largest at Pz and was particularly manifest in controls and guessers. It is interesting that the effect of the successfulness of inhibition on the P550 has also been established by Overtom et al. (2002) and Kok (personal communication, December 7, 2001). They related the enlargement of the P550 (at Pz) in the failed inhibition condition to what Falkenstein et al. (1991) have termed error positivity (Pe). The Pe is thought to reflect the operation of a conscious error-recognition system (e.g., Falkenstein et al., 1991, 1995; Rösler, 1995). Presumably, it is elicited when a participant thoroughly evaluates an incorrect response.

Assuming that our P550 reflects some sort of evaluation of the participants own performance, the pattern of P550 data suggests that the guessers are conscious of, and appraise, the errors they make. Accordingly, it may be concluded guessers find difficulty in inhibiting ongoing behaviors despite an adequate evaluation of their performance. Clearly, the P550 findings need replication for interpreting their full significance.

Is There an Association Between the Guessing Type of Dyslexia and ADHD?

van der Schoot et al. (2000) concluded that guessers can be compared with children with ADHD in that both clinical groups may suffer from the same type of deficits in executive functioning. They based this conclusion on the finding that guessers performed more poorly than spellers on the stop task, the Stroop task, and the Tower of London task, as well as on the finding that guessers displayed higher rating scores on the ACTRS. In children with ADHD, executive deficits have been shown by, for example, Barkley (1997), Pennington and Ozonoff (1996), and Oosterlaan and Sergeant (1998). These deficits have typically been associated with dysfunctions in the frontal and fronto-central brain areas (Barkley, Grodzinsky, & DuPaul, 1992; Grodzinsky & Diamond, 1992; Overtom, 1998). This study shows that the guessers' poor performance on the stop task (see van der Schoot et al., 2000) may be linked with a fronto-central deficit as well

(previously described). This suggests that guessers resemble children with ADHD not only at the behavioral level but also at the electrophysiological level. Accordingly, this study provides additional support for the idea put forward by van der Schoot et al. (2000), that there is a comorbid association between the guessing subtype of dyslexia and ADHD. Future research including a group of children with ADHD (and a comorbid RD+ADHD group) is needed to address the issue of (the nature of) the comorbidity of the guessing subtype of dyslexia and ADHD more directly.

Is There a Link Between the Guessers' Fronto-central Inhibitory Deficits and Their Reading Disturbance?

The crucial question that needs to be addressed is whether the executive-type deficiencies observed in guessers may also underlie their impulsive reading behaviors or that primarily a language-based disorder has to be assumed. Because this task did not tap critical elements of reading, no direct relationship between executive dysfunctions and specific reading disturbances can be deduced from this experiment. However, the finding that fast-inaccurate readers (i.e., guessers) can be differentiated from slow-accurate readers (i.e., spellers) and normal readers on a number of EF tasks (see van der Schoot et al., 2000), as well as the finding that the guessers' impairment in executive functioning is apparent not only behaviorally but also electrophysiologically, suggest that there is at least some type of association between them.

About the possible relationship between executive dysfunctions—in particular inhibitory deficits—and reading disturbances one can only speculate at this moment. van der Schoot et al. (2000) attempted to incorporate the concept of executive control into models of lexical activation (Morton, 1979; Morton & Patterson, 1980; Treisman, 1960) to explain the guessers' fast and inaccurate reading style. According to activation models, orthographic information about a target word accumulates gradually in the visual system, and as it accumulates, intermediary candidate words are concurrently primed or activated in the lexicon. The basic mechanism of word recognition is then to raise the activation level of one of the candidate words; that is, the target word, above some critical threshold value. van der Schoot et al. (2000) suggested that guessers may have generally lower word thresholds or may have more difficulty “dampening” the activation of candidates that are likely to be false. Both assumptions predict that in guessers, false candidate words have an increased chance of being prematurely identified as the target word. In a reading (aloud) task, this would be evidenced by an impulsive style of reading that is characterized by a high prevalence of substantive errors. It should be stressed that the account mentioned earlier of the guessers' impulsive reading style is highly speculative, and that more research is evidently needed to empirically establish

whether executive dysfunctions affect word recognition and reading style, and if so, in what way.

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